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Angiographic lesion characteristics can predict adverse outcomes after carotid artery stenting

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Background: **Carotid artery angioplasty and stenting (CAS) is an evolving and increasingly common endovascular treatment for carotid artery stenosis. Risk factors associated with an increased incidence of adverse periprocedural neurologic outcomes are being recognized. The goal of this study was to determine if certain angiographic lesion characteristics were predictive of higher risks of adverse outcomes.**

Methods: **A total of 421 patients who underwent 429 carotid artery stenting procedures between June 1996 and June 2005 for symptomatic or asymptomatic carotid stenosis, and in whom preoperative carotid angiograms and follow-up records were available for review, were selected from a prospectively maintained database. Demographic data and procedural variables were recorded, including the presence or absence of the use of a cerebral protection device. Angiograms were reviewed for the following carotid lesion characteristics: length of lesion, percentage of stenosis, ostial involvement, lesion ulceration, calcification, and presence of contralateral carotid occlusion. Periprocedural stroke and 30-day adverse event rates (stroke, myocardial infarction, and death) were recorded for each patient.**

Results: **The periprocedural all-stroke rate was 3.7%. Octogenarians had a higher incidence of 30-day adverse events at 10.0% vs 3.8% ($P = .029$). The incidence of periprocedural stroke was increased in lesions ≥15 mm long, at 17.0% (8 of 47) vs 2.1% (8 of 382; $P < .001$), and in ostial centered lesions, 7.1% (11 of 154) vs 1.8% (5 of 275; $P = .007$). Multivariate regression also identified these two variables as independently associated with 30-day stroke rate: lesion length ≥15 mm (odds ratio [OR], 6.38; 95% confidence interval [CI], 35 to 17.29) or ostial involvement (OR, 3.12; 95% CI, 3.12 to 8.36). Other variables, including lesion calcification, ulceration, degree of stenosis, or presence of contralateral occlusion, were not associated with adverse outcomes. When studied separately, the use of cerebral protection devices in 241 patients (56%) did not change our observed correlations between angiographic characteristics and adverse procedural events.**

Conclusions: **Certain lesion characteristics on angiography, such as length and ostial location, can predict adverse outcomes. The indication for CAS should be carefully evaluated in these cases. (J Vasc Surg 2008;47:81-7.)**

An abundance of evidence shows the benefit of relieving high-grade symptomatic and asymptomatic carotid artery stenosis by carotid endarterectomy (CEA).[1-5] More recently, carotid angioplasty and stenting (CAS) has been presented as an alternative to CEA for the treatment of carotid artery stenosis.[6-13] Both can be associated with complications, most notably, temporary or permanent neurologic deficits. Most of these events are related to thrombosis, distal embolization, or hemodynamic compromise. Factors associated with a higher incidence of complications for both procedures are progressively being described.

High-risk categories for CEA have gained widespread recognition, but only recently has attention focused on the identification of patients who are at greater risk for adverse outcomes during and after CAS. Only one clear limitation has become evident so far, namely, the higher rate of adverse outcomes in patients ≥80 years old. The Carotid Revascularization Endarterectomy versus Stenting Trial (CREST) lead-in phase and other studies have shown increased rates for stroke and stroke and death in octogenarians.[14,15] The increase in adverse outcomes seen with this population may be at least partly related to anatomic factors such as adverse arch anatomy and vessel tortuosity.[16] Other anatomic and patient characteristics may be associated with adverse outcomes but have not yet been fully described.

The composition of the target lesion has been found to be a predictor of stroke after CAS.[17] Less is known, however, about the anatomic characteristics and their association with outcomes. The goal of this study was to evaluate our database of CAS patients to determine if certain angiographic lesion characteristics were predictive of higher risks of adverse outcomes. This will help standardize the characterization of patients considered for CAS and help plan strategies to reduce complications.

METHODS

This study was approved by the Institutional Review Board of the University of Pittsburgh.

Patient population. From June 1996 to June 2005, >950 patients underwent CAS at Shadyside Hospital of the University of Pittsburgh Medical Center. Neurologists evaluated the patients before and ≤24 hours after the CAS procedure. A total of 429 CAS procedures met the minimum requirements for the review, which included (1)

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available angiographic data with a minimum of two different views of the carotid lesion, and (2) available follow-up records that included a preprocedural, an intraprocedural, an immediate postprocedural evaluation, and a 30-day follow-up visit.

Carotid artery stent procedure. Patients underwent CAS in the angiography suite by one of three interventionalists. All procedures were performed under local anesthesia with intravenous sedation. Aortic arch and cerebral angiography was performed before the stenting procedure. During the first year of patient accrual, patients were treated preprocedurally with ticlopidine (250 mg, twice daily). In the ensuing years of the study, patients received a 300-mg loading dose of clopidogrel and a maintenance dose of 75 mg daily for a minimum of 3 months. Aspirin was initiated only if indicated in the regulatory trial in which the patient was enrolled or by discretion of the interventionalist. No changes were made to a patient's regimen if he or she was already taking aspirin. During stenting, patients received anticoagulation with heparin to a target activated clotting time of ≥275 seconds.

Several stent systems were used, depending on the regulatory trial in which patients were enrolled, and included Precise in 80 (Cordis Endovascular, Miami Lakes, Fla), Smart in 106 (Cordis Endovascular), NextStent in 46 (EndoTex Interventional Systems, Cupertino, Calif), Acculink in 160 (Abbott Vascular, Santa Clara, Calif), and Wallstent in 32 (Boston Scientific, Natick, Mass). Embolic protection devices were coupled with the stents as systems according to regulatory trial protocols and included Angioguard (Cordis Endovascular), Accunet (Abbott Vascular), PercuSurge (Medtronic, Minneapolis, Minn), and Epi FilterWire (Boston Scientific). The use of cerebral protection became standard by July 2000. In patients not participating in a trial, stents and protection devices were inserted at the discretion of the interventionalist. The study excluded three patients who received balloon expandable stents.

Definitions. Stroke or cerebrovascular accident (CVA) was defined as a neurologic deficit lasting at least 24 hours. No distinction was made between minor and major strokes, and all ipsilateral and contralateral strokes were included. Transient ischemic attack (TIA) was defined as focal retinal or cerebral event from which there was no neurologic sequelae after 24 hours. Death was qualified as either a direct sequelae of stroke or from another cause.

Evaluation of angiograms. Digital subtraction angiographic images were collected retrospectively from the digital tape storage system of our angiography unit (General Electric, Milwaukee, Wis), from hard-copy angiography films, or from our digital radiologic archive (Stentor/Philips Medical Systems, Foster City, Calif). Ipsilateral and contralateral diagnostic images of the extracranial carotid arteries and the intracranial circulation were obtained before carotid stent placement. Images of the stented artery and of the intracranial circulation were also obtained after stent placement. The digital images of the extracranial carotid arteries were evaluated on a digital workstation with digital calipers. Hard-copy films of digital subtraction images were evaluated with a hand-held digital caliper (Absolute Digimatic, Mitutoyo USA, Aurora Ill). One investigator (S. S.) performed all of the measurements and was blinded to patient clinical information and outcomes. All measurements were digitally standardized to the known diameter of a guiding sheath or other standard internal control.

Angiographic lesion characteristics were recorded and defined as follows:

- *Lesion length* was defined in accordance with the American Heart Association/American College of Cardiology classification as modified by Ellis, et al.[18] Briefly, defined as the distance from the definite proximal to distal shoulder of the lesion in the projection that best elongated the stenosis. Only the portion of stenosis that was ≥ 50% was quantified.
- *Stenosis* diameter was determined by the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria, with the distal internal carotid serving as the reference segment. The most severe stenosis from a minimum of two projections was reported.
- *Ostial involvement* was indicated by a stenosis that was ostially centered; that is, the *maximal point* of stenosis was located at the internal carotid ostium.
- *Ulceration* of the plaque was noted if the plaque fulfilled radiographic criteria of ulcer niche, seen in profile as a crater from the lumen into a stenotic plaque and (when visible) a double density on face view.[19,20]
- *Calcification* was defined as radiologic densities readily seen within the apparent vascular wall of the artery at the site of the stenosis.[18]
- *Contralateral carotid occlusion* was defined as 100% occlusion of the contralateral common carotid or internal carotid arteries.

Data collection. Clinical, demographic, and stenting data were prospectively recorded on standard forms and entered into a database. Clinical and demographic data included age, sex, presence of symptoms in the last 60 days, and prior CEA. All periprocedural (≤24 hours) events were recorded. Primary clinical end points included stroke, death, or myocardial infarction ≤30 days of procedure. Patients were considered symptomatic if they had TIAs, amaurosis fugax, or stroke with clinically correlated, lateralizing symptoms ≤60 days preceding the carotid intervention. Outside of this window, patients were considered asymptomatic.

Morphologic data were recorded retrospectively by reviewing angiographic films. The morphologic variables studied were lesion length, ostial involvement, calcification, plaque ulceration, and lesion severity. The presence of contralateral carotid artery occlusion was also recorded. Procedural variables were recorded and included the presence or absence of the use of a cerebral protection device.

Statistical analysis. Periprocedural and 30-day adverse event rates by demographic, angiographic, and device characteristics were summarized as percentages. Statistical

comparisons were made using either the χ^2 test or the Fisher exact test. The Cochran-Mantel-Haenszel statistic was used to evaluate trends in adverse event rates and lesion length categories. Logistic regression was used to determine variables associated with 30-day stroke, and the risk is represented by the odds ratio (OR). Logistic regression was used to initially screen all clinical variables for univariate association with 30-day outcomes of interest at $P < .15$. Individual variables identified were then assessed in a forward stepwise manner using a criterion of $P < .05$. The final model included all significant variables and any other variables thought to be relevant (eg, age). Goodness-of-fit was assessed using the Hosmer-Lemeshow method, and all models were considered to be adequate ($P > .05$).

RESULTS

Demographic, clinical, and morphologic characteristics. A total of 429 carotid arteries in the 421 patients were treated during 429 procedures. In five of the 429 procedures (1.2%), carotid stenting was aborted before stent insertion secondary to various technical complexities, and no procedural or postprocedural morbidity developed in these patients. A recorded evaluation by a neurologist was available in 96% of patients. The mean age of the patients was 72.3 ± 8.7 years: 144 (34.2%) were <70, 188 patients (44.7%) were 70 to 79, and 89 (21.1%) were ≥80. Men comprised 61.5% of the study population.

Univariate analysis was used to evaluate patient characteristics and comorbidities, including gender, coronary artery disease, congestive heart failure, hypertension, diabetes mellitus, prior myocardial infarction, chronic obstructive pulmonary disease, smoking, and hypercholesterolemia for association with 30-day major adverse events of stroke, myocardial infarction, and death. No relationship was found, and these results have previously been published.[15] Asymptomatic carotid artery stenosis was present in 65% of patients. Prior ipsilateral CEAs were present in 134 patients (32.1%). Cerebral protection devices were used in 241of the total procedures (56.2%).

Procedural outcomes. A periprocedural stroke occurred in 16 patients (3.7%), of which 15 were ipsilateral and one was contralateral. An ipsilateral stroke developed in an additional three patients after the periprocedural period but ≤30 days (days 1, 14, and 23); as a result, the 30-day stroke rate for this study was 4.4%. Myocardial infarction occurred periprocedurally in one patient (0.2%) and ≤30 days in another patient. Two patients (0.47%) died periprocedurally, and an additional six patients died ≤30 days. Seven of these eight patients (88%) had a periprocedural stroke and died from hemorrhagic conversion of their infarct (n = 2) or from other sequelae of their periprocedural infarct; thus, most deaths were stroke related.

The overall periprocedural adverse event (stroke, death, or myocardial infarction) rate was 3.96%, and the 30-day adverse event rate was 5.1% (Table I). The difference in the 30-day stroke rate between asymptomatic (3.9%) and symptomatic patients (5.3%) was not significant ($P = .623$).

Table I. Procedural outcomes

Outcome	*Periprocedural (%)*	*30-day (%)*
Stroke	3.7	4.4
Transient ischemic attack	3.0	3.3
MI	0.23	0.47
Death	0.47	1.9
Adverse event (stroke, death, MI)	3.96	5.1

MI, Myocardial infarction.

Octogenarians had more adverse events than younger patients (Table II). The incidence of perioperative stroke was 2.6% in patients <80 years and 7.8% in those ≥80 ($P = .053$). The incidence of 30-day death also paralleled this difference: 0.9% for younger patients and 5.6% for octogenarians ($P = .012$). When grouping major adverse events (stroke, death, or myocardial infarction), the incidence was 3.8% in younger patients and 10.0% in octogenarians ($P = .029$).

Cerebral protection was used in 241of the 429 procedures (56.2%). The incidence of perioperative stroke in patients without cerebral protection was 4.8% vs 2.9% in those who had CAS with a cerebral protection device ($P = .32$). The incidence of 30-day stroke was also similar at 5.8% vs 3.3 ($P = .24$). Five different stent systems were used during this study, and there were no differences in outcomes depending on system type.

Morphologic characteristics and postprocedural events. Angiographic lesion characteristics and associated outcomes are summarized in Table II. Lesion calcification, ulceration, and degree of stenosis were not significantly associated with neurologic complications. The presence of contralateral occlusion was also not significantly associated with periprocedural adverse events.

The median ± SD length of carotid lesions was 8.23 ± 5.2 mm (range, 1.00 to 31.05 mm): 95 lesions (22.1%) were <5.0 mm, 182 (42.4%) were 5 to 10 mm, 105 (24.5%) were 10 to 15 mm, and 47 (11.0 %) were ≥15 mm. Lesions ≥15 mm were associated with higher risk of adverse outcomes. The incidence of periprocedural stroke was 2.1% in patients treated with lesions <15 mm compared with 17.0% in patients with a treated lesion ≥15 mm ($P < .001$). Patients with treated long lesions were also more likely to experience 30-day adverse events: <15 mm, 3.4% vs ≥15 mm, 19.1% ($P < .001$).

No trend in the risk of adverse events was noted in patients with treated lesions <15 mm (P trend ≥.80 for all comparisons). The incidence of periprocedural stroke in patients with lesions <5.0 mm, 5 to 10 mm, and 10 to 15 mm was, respectively, 2.1%, 2.2%, and 1.9% (overall $P = .99$), and the respective incidence of adverse events ≤30 days was 3.2%, 3.3%, and 3.8% (overall $P = .96$).

Lesions in which the maximal point of stenosis was located at the internal carotid ostia were seen angiographically in 154 of the vessels (35.9%) that were studied. The incidence of periprocedural stroke was 7.1% in patients with

Table II. Correlation of clinical and angiographic features with postprocedural events

Variable	*Patients, No.*	*Periprocedural stroke (%)*	*P*	*30-day stroke (%)*	*P*	*30-day adverse event (%)*	*P*
Age ≥ 80			.53		.037		.029
No	339	2.6		3.2		3.8	
Yes	90	7.8		8.9		10	
Ulceration			.59		.62		.82
No	286	4.2		4.9		4.9	
Yes	143	2.8		3.5		5.6	
Calcification			.8		.35		.18
No	185	3.2		3.2		3.2	
Yes	244	4.1		5.3		6.6	
Ostial centered			.007		.015		.01
No	275	1.8		2.5		2.9	
Yes	154	7.1		7.8		9.1	
Lesion length ≥15 mm			<.001		.002		<.001
No	382	2.1		2.9		3.4	
Yes	47	17		17		19.1	
Stenosis, %			.72		.75		.5
≤59	76	1.3		3.9		5.3	
60-69	102	5.9		6.9		7.8	
70-79	120	2.5		2.5		2.5	
80-89	81	4.9		4.9		6.2	
≥90	50	4		4		4	
Contralateral occlusion			.71		.49		.34
No	371	4		4.8		5.7	
Yes	58	1.7		1.7		1.7	
Cerebral protection			.32		.24		.076
No	188	4.8		5.8		7.4	
Yes	241	2.9		3.3		3.3	

Table III. Independent risk factors associated with 30-day stroke using logistic regression

Factor	*OR*	*95% CI*	*P*
Age ≥80	2.49	0.93-6.67	.069
Ostial-centered lesion	3.12	1.17-8.36	.023
Lesion ≥15 mm	6.38	2.35-17.29	<.001

OR, Odds ratio; *CI,* confidence interval.

Table IV. Interaction between age and lesion length or ostial involvement associated with 30-day stroke using logistic regression

Variable	*OR*	*95% CI*	*P*
Age and lesion length vs <80 y and <15 mm			
Age ≥80 y and lesion <15 mm	3.36	0.99-11.42	.052
Age <80 y and lesion ≥15 mm	8.77	2.47-31.10	<.001
Age ≥80 y and lesion ≥15 mm	13.12	2.81-61.31	.001
Ostial-centered lesion	3.09	1.16-8.25	.024

treated lesions with ostial centering vs 1.8% in patients with treated lesions without ostial centering ($P = .007$). Rates of 30-day adverse events were also greater in patients with ostial lesions (9.1% vs 2.9%; $P = .01$).

Multivariable logistic regression identified three main effects as being associated with 30-day stroke (Table III): age ≥80, ostial lesions, and lesion length ≥15 mm. The strongest predictor was lesion length ≥15 mm (OR, 6.38; 95% CI, 2.35 to 17.29), followed by the treatment of a lesion in the ostium (OR, 3.12; 95% CI, 3.12 to 8.36). Age also was associated with a higher risk of stroke; however, statistical significance was not met at the $P = .05$ level (OR, 2.49; 95% CI, 0.93 to 6.67; $P = .069$).

To evaluate the interactions between octogenarian status and ostial involvement or lesion length, multivariate analysis was performed to evaluate for a confounding effect (Table IV). In this analysis, the comparison group is patients <80 years old with a lesion <15 mm. Even though the greatest risk of stroke was in the subgroup of patients who were ≥80 years *and* who had long lesions (OR, 13.12; 95% CI, 2.81 to 61.31), a long lesion was also a predictor of stroke amongst nonoctogenarians (OR, 8.77; 95% CI, 2.47 to 31.10). Ostial-centered lesions also were a significant risk factor independent of octogenarian status.

The routine of use of cerebral protection devices started in July 2000 for this study group, and thus the devices were used in 241 of the 429 procedures. To study if the routine use of a cerebral protection device had any impact on the correlation between angiographic characteristics and procedural events during CAS, this subpopulation was studied separately (Table V). Lesions ≥15 mm were associated with increased risk of 30-day stroke vs lesions <15 mm (16.7% vs 1.84%, $P = .004$). Increases in 30-day stroke rates were

Table V. Correlation of angiographic features with postprocedural events in patients with cerebral protection

Feature	*30-day adverse event (%)*	*P*
Ulceration		.71
No	3	
Yes	4	
Calcification		1
No	3.1	
Yes	3.5	
Ostial centered		.01
No	0.7	
Yes	6.9	
Length ≥15 mm		.004
No	1.8	
Yes	16.7	
Stenosis, %		.28
≤59	5.9	
60-69	2.4	
70-79	0	
80-89	4.9	
≥90	4.8	
Contralateral occlusion		1
No	3.6	
Yes	2.1	

Table VI. Independent risk factors associated with 30-day adverse event using logistic regression in patients with cerebral protection

Factor	*OR*	*95% CI*	*P*
Ostial-centered Lesion	9.38	1.10-80.29	.041
Lesion ≥15 mm	9.66	2.11-44.32	.003

OR, Odds ratio; *CI,* confidence interval.

also seen with patients with ostial (6.9%) vs nonostial (0.7%) involvement ($P = .01$). Logistic regression showed ostial involvement and a lesion ≥15 mm were strong predictors of adverse outcome, even in the subgroup of patients who received cerebral protection during CAS (Table VI).

DISCUSSION

The quickly evolving technique of CAS, which is an alluring and minimally invasive alternative to CEA, is being analyzed in a number of centers around the world. The primary objective of this study was to identify morphologic lesion characteristics that may affect the procedural risk of CAS. Although advanced age was a significant predictor of complications in our study, some anatomic lesion characteristics were independent predictors of adverse neurologic event rates.

We identified two angiographic lesion risk factors significantly associated with periprocedural neurologic deficits and adverse outcomes: the presence of long stenotic lesions (≥15 mm) and involvement of the internal carotid ostium. Our median lesion length was 8.2 mm, yet a number of the patients in our study population had long lesions that were ≥15 mm. These individuals had an alarming rate of periprocedural stroke (17.0%) and 30-day adverse events (19.1%).

No comments can be made about the composition of these long plaques and whether they inherently differ from shorter lesions. Longer lesions, however, have a larger atherosclerotic "plaque burden" and hence conceivably carry a higher risk that particles will be dislodged during balloon angioplasty and stenting. Longer lesions also may require longer stents and, at times, multiple angioplasties to ensure adequate stent dilation across a stenosis.

Highly stenotic lesions were not associated with increases in stroke rates or adverse outcomes. Lesions with ≥90% stenosis had similar adverse neurologic events as lesions that were less stenotic. One would contemplate that maneuvering beyond a tight stenosis would be more precarious than maneuvering through a stenosis of lesser degree and would thus be associated with a greater number of unfavorable neurologic events; however, our finding was consistent with findings in other studies.[21,22] It is possible that lesion length is a better predictor of adverse outcome with CAS than the percentage of stenosis simply because it correlates better with a plaque's "exposed surface area" and hence more plaque burden.

Lesions in which the maximal point of stenosis was located at the internal carotid ostium were also associated with an increase in periprocedural stroke of 7.1% vs 1.8%. Some of these lesions, despite having a point of maximal stenosis elsewhere in the internal carotid artery, would "extend" to the ostium. We, however, limited the definition of ostial involvement to include only lesions that had their *maximal* stenosis at the internal carotid artery ostia.

The reason for the observed increased risk with ostial lesions may be multifactorial. It is possible that ostial lesions simply are more difficult to initially engage with a wire compared with more distal lesions, and as a result have an increased risk of triggering embolic debris. Another mechanism may be hemodynamic instability (hypotension, bradycardia, transient asystole, or a combination) associated with carotid sinus stimulation, which has been shown to occur more often in patients with ostial lesions.[23] Such instability has been associated with stroke and cardiac complications.[24-26] Hence, it is plausible that our observed increase in neurologic events observed with ostial lesions during CAS may be related to more hemodynamic hyperreactivity during the procedure. Unfortunately, such detailed events were not recorded prospectively to allow further testing.

One limitation of our study was the long accrual span, during which many technical advances in CAS occurred, including the development of cerebral protection. Even though our patients who underwent CAS with cerebral protection did have lower rates of neurologic adverse events, these rates were not significant. This inability to demonstrate a significant difference may be due to small sample size (type II error).

Also of note, when our subgroup of patients who underwent CAS with cerebral protection was analyzed, those with ostial and long lesions continued to show sig-

nificantly increased stroke rates. Cerebral protection use is associated with increases in intervention time and may have a negative influence on embolization rates themselves.[27,28] It is possible that the maneuvering of filter devices through ostial or long lesions before their deployment may be a significant cause for the observed events associated with these lesions.

A second shortcoming was the exclusion of a large number of patients (n = 521) who lacked complete 30-day follow-up (n = 413) or the unavailability of complete angiographic films (n = 108). The sample size however remains high enough to allow the comparisons to be valid.

Although not well characterized, other anatomic characteristics may affect the incidence of adverse neurologic events after CAS, such as atherosclerotic arch disease, tortuosity, or origin of the great vessels.[29,30] These were not included in our analysis and may have eliminated some potential confounding variables. Unfortunately, many of these were not always accessible and would have eliminated many subjects from analysis. It is unlikely, however, that these variables would have changed our results significantly, because lesion length and ostial location have never been related to arch abnormalities.

The low incidence of event rates after CAS may have prevented the identification of other predictive factors of adverse outcomes. Evidence is mounting, however, that subclinical infarction may be much more common with CAS than previously suspected. A prospective analysis including diffusion-weighted magnetic resonance imaging may provide better correlation of adverse outcomes of CAS with lesion characteristics.[31,32] All events analyzed in our report were noted on clinical examination, which may underestimate the true incidence of embolization and the effect of certain anatomic characteristics. Because clinical events after CAS are fairly infrequent, additional surrogate markers for clinical stroke and silent cerebral ischemia that occur at greater frequencies would be of use.

Other lesion characteristics not identified on angiography, such as gray-scale median (GSM), may be as important as those identified in this study. Biasi et al[17] have found that a GSM <25, associated with echolucent plaques, predicts accurately a high incidence of neurologic events after CAS.[17] Characterizing lesions with virtual histology-assisted intravascular ultrasound imaging may also help to determine the compositions of ostial and long lesions and if they inherently differ from other lesions.

Our study suggests that a combination of risk factors may be additive in predicting complications. Once all factors such as arch anatomy, lesion characteristics, and physiologic factors such as age and symptoms are ultimately well defined, a high-risk patient category for CAS is likely to emerge that will permit better selection of cases. It would be interesting to evaluate other large existing CAS databases (Society for Vascular Surgery Registry and various European databases) for additional confirmation of our results. This is likely to identify contraindications for CAS and categories of patients with expected good outcomes.

CONCLUSION

It is evident that certain lesion characteristics on angiography, such as length and ostial location, can predict outcomes after CAS. Until further validation of these findings, caution and restraint should be exercised in stenting these lesions.

AUTHOR CONTRIBUTIONS

Conception and design: MM, MW
Analysis and interpretation: SS, MM
Data collection: SS, SFS
Writing the article: SS
Critical revision of the article: MM
Final approval of the article: MM
Statistical analysis: SS
Obtained funding: MM, MW
Overall responsibility: SS

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